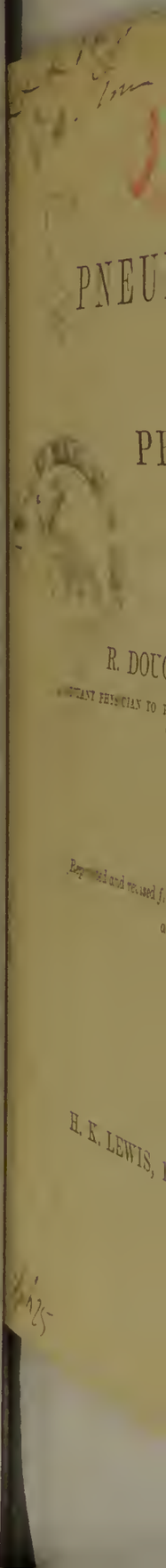

NOTES

ON THE

PNEUMOTHORAX

OCCURRING IN

PHTHISIS

BY

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NOTES ON THE PNEUMOTHORAX OCCURRING IN PHTHISIS.

The following observations refer only to pneumothorax occurring in cases of phthisis.

The tendency to pneumothorax in phthisis consists in the constant softening down of deposited tubercle and pneumonic infiltrations, which takes place around already-formed cavities, or leads to the formation of new ones; this tendency is most generally resisted by the thickening and induration of the pleura and lung tissue, and by the formation of firm adhesions between the pulmonary and costal pleuræ. In some cases, however, the softening process appears to advance too rapidly for the preservative morbid process to keep pace; the pleura becomes extensively undermined by the abrupt softening down of large masses of degenerated tubercle or pneumonia situated immediately under it; its vascular supply is interfered with, one or two small sloughs form, and on a slight exertion or cough one of these gives way, and pneumothorax results.

This is most commonly observed in cases of phthisis which are either of the pneumonic variety, or in which the epithelial pneumonia is far more extensive than the tubercular deposit. Out of 60 cases of Phthisis which I care-

fully examined post mortem last year, 12 were of the pneumonic variety, and of these, 3 had terminated in Pneumothorax, while of 23 cases of tolerably pure tubercular phthisis, only 2 terminated in Pneumothorax; also of 9 cases of tuberculo-pneumonic phthisis in which the tubercle and the pneumonia were in about equal proportions, 1 terminated in Pneumothorax, and in 8 cases of phthisis in which there was marked contraction of one lung (Fibroid Phthisis) there was no instance of perforation. The pleura becomes most strongly adherent and thickened in the last variety, least so in the first.

There appears also to be another way in which perforation may take not so common as that above mentioned, but which the writer of this paper has seen exemplified in two cases (one published in the *Medical Times and Gazette*, April, 1866, and Case 4)—viz., by the formation of a sinus leading from an old cavity, and resembling those seen in limbs leading from carious bone, or in the neighbourhood of chronic abscesses. A cavity in the lung after enlarging in the ordinary way to a certain extent, ceases to do so, and becomes converted into a chronic abscess, with vascular walls yielding pus; sinuses not unfrequently extend from cavities of this kind to the pleural surface, and, in a few unfortunate cases, where adhesions have not been previously formed, may open into the pleural cavity. Most commonly there are strong adhesions over a cavity of this kind. In Case 4, above quoted, on separating some firm adhesions nearer the apex of the lung, another opening was found, and corresponding to

it on the costal pleura was a deep erosion representing the blind end of a sinus which had penetrated through the two adherent layers of the pleura: such a sinus may penetrate through the chest wall. In the first case the cavity was a very old one, and the sinus opened through the posterior mediastinum into the opposite pleural cavity by a well-defined aperture, the tissue having been, "as it were, bored through by a circular canal" (loc. cit.).

The *symptoms* of pneumothorax are often completely masked, while, on the other hand, all the symptoms, sudden pain, dyspnœa, shock, etc., may be produced by the acute congestion stage of pneumonia with superficial pleurisy, supervening in a case of advanced phthisis. The principal symptoms are—sudden acute pain, followed by great dyspnœa and shock; pulse frequent, feeble, small; respiration relatively more frequent than the pulse; usually aphonia; occasionally great hyperæsthesia of the affected side; position of the patient frequently changed, or dorsal, with head raised and inclination to sound side (after effusion has taken place, to affected side), or the patient may support himself on his knees and elbows, with slight inclination to left and right side alternately. M. Gaide (a) explains the instinctive adoption of this position as allowing the heart to rest on the thoracic wall so as not to compress the sound lung. Orthopnœa, with slight inclination forwards, is a common

(a) "Obs. à l'Hôpital St. Antoine," *Archives Gén. de Medicine*, t. xvii. 1288.

position. If the opening be free, the patient may continue in the recumbent posture.

Physical Signs.—These also simply require enumeration, with the exception of two or three. Bulging of the affected side, with raising of the shoulder; effaced intercostal spaces; immobility or impaired mobility of the sibe. Displacement of heart towards the opposite side. Hyper-resonance on percussion to true tympanitic note. Respiration absent ,or very feeble, or amphoric at one or more points of the chest, with metallic whisper and metallic echo on cough (true pectoriloquy is rarely heard); metallic tinkling and vocal fremitus absent or diminished. Later, when effusion has taken place, there is dulness below, hyper-resonance above, the two shifting with the position of the patient; splash on succussion, intercostal fluctuation, etc. This fluctuation gives to the finger, on percussion at the line of contact of the air and fluid, a peculiar thrilling sensation.

Of all these signs the three important ones for diagnosis are—hyper-resonance; absence of, or feeble, respiration sounds, or amphoric breathing; displacement of heart.

These three signs are the most constant, and, being present, render the diagnosis certain. They can also be observed without distressing the patient unnecessarily during the period of shock. Laennec only mentions displacement of heart casually and as a possibility. M. Gaide was the first to insist upon it as a clinical fact. It is a fact of the first importance, and requires a few more words for its explanation.

The commonly received doctrine that the heart is

displaced towards the opposite side by the pressure of the escaped air is not wholly true, for the displacement is instantaneous. This was first noticed as a clinical fact by the writer in 1865, in a case seen within two or three minutes of seizure; the explanation was not then, however, apparent. The true mode of production seems to be the following:—The mediastinum (including the heart) is the only boundary common to the two thoracic cavities, which is flaccid, and admits of a certain degree of passive movement to either side. It is maintained in position (and with it the heart) by the opposing *elastic tensions* (a) of the two lungs, each tending to draw it (the mediastinum) to its own side. Now, if the elastic tension of one lung be destroyed by the admission of air from without into the pleural cavity, causing the lung to collapse, the elasticity of the other lung has no opposing force, and immediately draws the mediastinum, and with it the heart, to its own side. Moreover, when an inspiratory effort is made, it is impossible for the elasticity of the sound lung to be overcome, and, consequently, for its expansion to commence, until the mediastinum has become sufficiently convex to be *stretched*, and thus to form a resisting wall. In this way and to this extent the displacement of the heart is immediately and necessarily consequent upon the admission of air into the pleura. Beyond this it may be increased by the direct pressure produced by the subsequent accumulation of air.

(a) This tension was found by Dr. Salter, in some experiments on the dog, to be equal to from twelve to eighteen inches of water. Lecture at the College of Physicians, 1865.)

The following experiments were made to test the accuracy of this view:—

April 16, 1868.—Subject, a young woman in the post-mortem room, Middlesex Hospital. A skin flap was raised from the median line on the left side, and a long needle thrust vertically through the pericardium into the heart, at the fourth interspace, left border of sternum. The pleura was then opened by an incision at one of the left interspaces; a faint sucking noise was heard, and the needle was immediately deflected to the left. The arms were then drawn upwards equally, making a movement of inspiration, and the deflection of the needle was increased. On opening the chest no adhesions were found; there were some pyæmic deposits in lungs, and slight consolidation of both bases. This experiment was made in the presence and with the kind assistance of Mr. Henry Arnott. The diagrams below will illustrate this and the following experiment:—*

April 23.—A moderate-sized dog was put under chloroform, and a skin flap of a few inches raised from the præcordial region; the muscles were then divided by a circular incision of about two inches diameter. A long needle was then thrust into the heart at the left border of the sternum; it vibrated with the cardiac pulsation, and had a slight inclination to the left of the vertical line. At a moment when the respirations were regular and uniform, an opening was made into the left pleura through one of the lateral interspaces; the air was freely *sucked* in, and the needle instant-

* See next page.

Diagram of Section of Chest viewed from above.

FIG. 1.

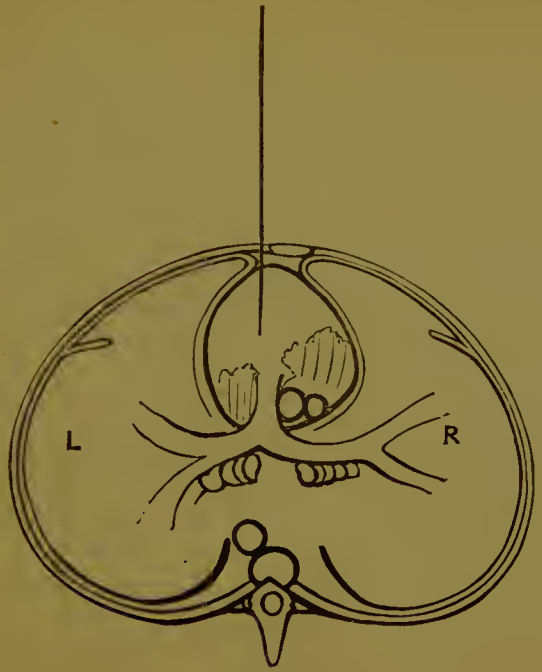


FIG. 1.—Before admitting air into left pleura.

FIG. 2.

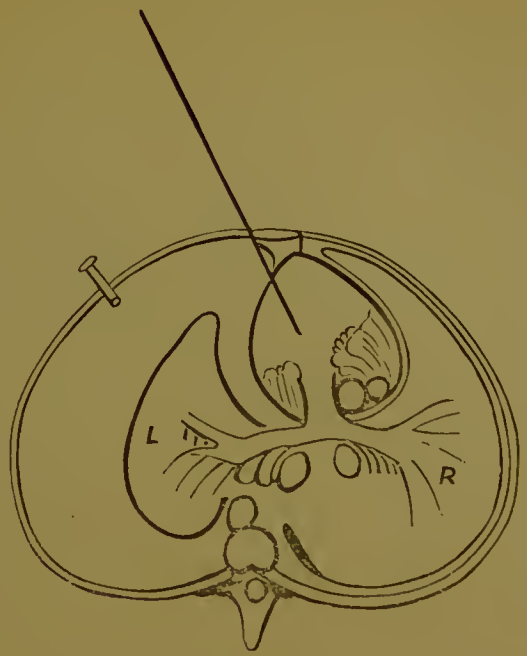


FIG. 2.—After entry of air into left pleura.

ly deflected strongly to the left. The total deflection was about 35° from the vertical line, the accidental deflection at first being perhaps 15°. This experiment was made with the kind assistance of the late Mr. Alexander Bruce, M.S. The deflection of the needle was, of course, in both these cases an index of the displacement of the heart.

The two following cases, reports of which were inserted in the Medical Times and Gazette for Aug. 21st 1869, seem to confirm the correctness of the above observations and experiments, and I venture to reprint them.

Case 1.—Walter C., aged 21, admitted into the Brompton Hospital, under the care of Dr. Cotton, February 12th 1869. Patient had a strong hereditary predisposition to phthisis and dated his present illness from thirteen months previous to admission. On January 10th while at the Chelsea House, awaiting his turn for admission, he was seized with pain in the left side. He was seen on Jan. 12 by Mr. Charles Joubert, resident Clinical Assistant at the Hospital, who found the whole left front of the chest tympanitic on percussion, with distinct amphoric respiration, most plainly audible below the clavicle. The hyperresonance did not extend beyond the mid-axillary vertical line; there was dulness posteriorly with scattered humid crepitation. The heart's impulse was seen, and felt at the fifth right intercostal space within the nipple; the patient had remarked at the time of his seizure that the heart beat to the right of the sternum. Decubitus right; respirations thirty in the minute; pulse 100. There were signs of excavation and softening at the right apex.

Jan. 15. Better; no urgent symptoms of pneumothorax.

On admission into the Hospital (February 12th), the hyperresonance extended beyond the median line in front, the amphoric respiration was well marked in front and laterally to the base, and metallic echo and tinkle were audible. The

deficient resonance and moist râles were still present posteriorly. The disease in the opposite lung had advanced. The heart's maximum impulse was to the right of the sternum; the apex was however, ascertained by percussion and palpation to be at the ensiform cartilage. The respirations were 30 in the minute, the pulse 96. No urgent dyspnœa. The patient was weaker and lingered on, gradually sinking from the progress of the general disease, without any material change in the physical signs. He died May 26th, 1869.

Autopsy thirty hours after death.—No difference noticeable in the relative size of the two sides of the chest. A trocar and canula connected by tubing with a water pressure-gauge, was inserted at the fifth left interspace, to ascertain the air-pressure within the pleura. This was found to be *nil*. A stilette was then thrust in at the fourth right interspace near the sternum, the trocar withdrawn, and the cartilages removed in the ordinary way, the heart being transfixed in position by the stilette. The exact position of the heart was as follows: The apex was behind the sternum, and slightly to the left of the median line—*i.e.*, in the verticle line of the left sterno-clavicular articulation, and at the level of the fifth rib. The left border of the heart occupied the median line, with a slight inclination to the left; the right border was touched by a line drawn vertically from the middle of the right clavicle. The left pleura contained a small quantity of purulent fluid; the lung was collapsed backwards, and a large opening capable of admitting the end of the little finger was seen near the apex, through which air bubbled freely on blowing into the trachea. The right lung was excavated at the apex, it was partially collapsed below, and presented scattered patches of grey tubercle and pneumonia.

Case 2.—Martha B., aged 19, admitted into the Brompton Hospital under the care of Dr. Alison, March 29th, 1869. This patient was hereditarily predisposed to phthisis, and had suffered from cough for six months.

On admission she presented signs of cavities at both apices, with softening below, the left lung being more diseased than the right. The symptoms of pneumothorax came on insidiously, but on May 8th, the signs were distinct on the left side, the amphoric respiration being very well marked. The patient died May 21st.

Post Mortem.—There was no difference in the expansion of the two sides; the left was hyperresonant, the resonance extending over the ordinary position of cardiac dulness, and across the median line to the right margin of the sternum.

The air-pressure within the pleura was tested as in the preceding case and found to be *nil*. The heart was then transfixed by a stilette thrust in at the fourth right interspace close to the sternum, and the cartilages removed. The mediastinum was found to be curved with its convexity to the right; thus—commencing at the episternal notch, its left border arched to the right border of the sternum opposite the third cartilage, and thence gradually downwards to the left of the ensiform cartilage. The apex of the heart was opposite the fifth rib, exactly in the middle line; its right border corresponded with a line drawn vertically downwards from the middle of the right clavicle; the heart's axis was more vertical than natural. The left pleura contained about a pint of purulent fluid. There was a large opening in the lung pleura opposite the third rib, freely communicating with a cavity.

Both lungs were extensively disorganized, the disease being the pneumonic variety of phthisis. On the surface of the left pleura there were many scattered yellow patches, where the pleura had nearly given way from rapid softening down of subjacent lung consolidations.

These cases shew very clearly the great displacement of heart which may occur without any direct pressure, as the simple result of the elastic recoil of the unruptured lung, acting upon the flaccid mediastinum, unopposed by that

of the opposite lung. This physiological fact places cardiac displacement in the first rank among the signs of pneumothorax, since it necessarily follows immediately upon the entry of air into one pleura. These cases further shew an important clinical fact—viz., that displacement of heart, even when very considerable, is not necessarily a sign of pressure, and is therefore of itself no sufficient reason for the performance of paracentesis.

The heart may beat so feebly as to be distinguished only with great difficulty, in which case its position may be ascertained by means of the stethoscope, or the displaced cardiac dulness may be detected by percussion. In one case of Dr. Quain's, at the Brompton Hospital, there was no cardiac dulness to be found, nor any impulse, and only with difficulty could any sound be detected. In this exceptional case an opening in the pericardium allowed the air to escape from the pleura into it, and the heart was compressed backwards.

If there be well-marked amphoric respiration, the communication between the pleura and bronchial tubes will be free, and the opening patent. If there be no amphoric respiration, or complete absence of respiration, the bronchial tubes communicating with the cavity are very small, or the aperture in the pleura is valvular, the prognosis derived from this negative sign being very grave, and confirmed by the steadily increasing dyspnœa.

The intensity of the symptoms in pneumothorax is influenced by—

A. The condition of the lung previous to its rupture. If the lung be already extensively diseased, the symptoms produced by its rupture will be much less marked than if it were comparatively healthy. The two points to be considered under this head as affecting the severity of the symptoms are—

(1) The greater or less amount of respiratory area suddenly cut off.

(2) The increased resistance to the flow of blood through the capillaries of the affected lung.

The former bears a very important part in the production of the dyspnœa, and depends directly upon the amount of healthy tissue left in the lung which has suddenly become collapsed; the greater the amount the greater the disturbance of the equilibrium, if one may use the term, between the aërating power and the blood volume to be aërated, and hence the more urgent the dyspnœa.

But while this has long been known and appreciated, is there not also another agent in the production of the dyspnœa—viz., the pressure to which the lung is exposed and the consequent impediment to the circulation through it? It was proved by Goodwyn in 1788, and more lately by Mr. Erichsen in 1845, that mere collapse of the lung does not materially affect the circulation through it, and Mr. Erichsen's experiments tend to show that the cessation of the vital changes of respiration does not retard the circulation through the lung. But in pneumothorax, at least in some cases, the lung is subjected to considerable pressure, and is more completely collapsed than in any of these experiments.

I have ascertained post mortem, the actual pressure to which the lung is subjected in pneumothorax in ten cases. The apparatus used for this purpose is simple enough. It consists of a trocar with well-fitting canula, having a lateral branch, which is connected by india-rubber tubing with a water pressure-gauge; the water may be conveniently coloured with a drop of Condy's fluid. The trocar, with canula, is thrust into the chest, the trocar withdrawn, and the height to which the fluid rises noted.

Case 1.—February 15, 1867: 30 hours after death. Pressure=4 inches of water.

Case 2.—June 20, 1867; (attack June 18) 12 hours after death. Pressure=5 $\frac{3}{10}$ inches of water.

Case 3.—July 15, 1867; about 14 hours after death. Pressure=2 inches of water.

Case 4.—August 21, 1867; 16 hours after death (seizure 18th morning; death 20th evening. Pressure=3¾ inches of water.

Case 5.—October 8, 1867; 30 hours after death (seizure 6th. 12·30 a.m.; death 7th, in morning). Pressure=2 inches of water.

Case 6.—April 1, 1868; 30 hours after death. Pressure=4 inches of water.

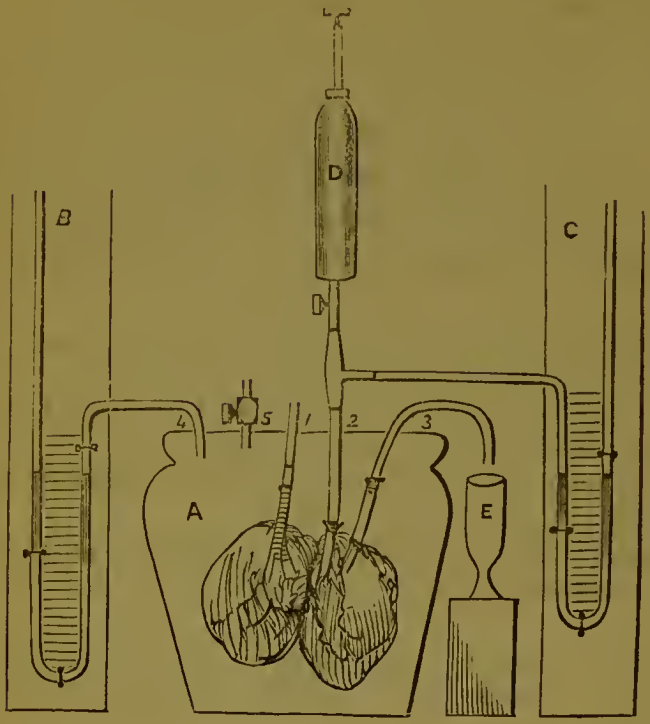
Case 7.—May 15, 1868; about 20 hours after death. Pressure=1¼ inches of water.

Case 8.—July 2, 1868; 24 hours after death. Double pneumothorax. Pressure left side (old)=3½ inches of water. Pressure right side (recent; 10 minutes)=2·7 inches of water.

Two other cases are related above in which the pressure was nil.

The following sets of experiments had been previously

undertaken to ascertain what effect the compression of a lung by air would have upon the circulation through it:—



For this purpose an apparatus was fitted up, consisting of a large glass jar, A, across the mouth of which is stretched a piece of bladder perforated by five tubes accurately fitted, so that the whole when secured, is air-tight. To tho central tube, 1, the lung is attached by the trachea, and to its other end a short piece of gutta-percha tubing is attached. To the next tube, 2, the pulmonary artery is secured, and the free end is left open for the insertion of the nozzle of an injecting syringe. The third tube is connected

with the pulmonary vein, and its unattached end is prolonged by tubing, so as to reach over the side of the jar into a glass measure, E. The fourth tube communicated by one end with the interior of the jar, and by the other with a pressure gauge, B. to note the air-pressure within the jar. The fifth tube, also free within the jar, is provided at its other end with a stopcock, so that air can be injected and retained at any pressure required. The syringe has its nozzle fitted into a T-shaped tube, which is connected by one branch with the pulmonary artery tube, 2, and by the other with a hæmadynamometer, C, which measures the injecting force employed.

The first set of experiments were made January 11, 1867, with the kind assistance of the late Mr. Alexander Bruce and Dr. Green. The lungs used were those of a recently killed dog. They were attached by the trachea, common pulmonary artery, and vein to the tubes 1, 2, and 3 respectively, and the whole apparatus carefully secured, Whipped bullock's blood was used for injection.

Experiment 1.—Lungs moderately expanded by inflation through tube 1; atmospheric pressure within the jar normal; blood injected into the pulmonary artery with a pressure of between 1½ and 1¾ inches of mercury. The blood flowed from the pulmonary vein in a fair equable stream and filled the vessel, E, up to a given mark in 50 seconds.

Experiment 2.—Ligature removed from the trachea, tube 1, and the lungs allowed to collapse by their own elasticity; atmospheric pressure within the jar still being normal. Injection again employed at the same pressure. It still took 50 seconds to fill the measure, E, to the same point.

Experiment 3.—Air was now forced into the jar by a syringe through the tube, 5, until the mercurial gauge, B, indicated a pressure of ¾ inch. The stopcock was then closed, and injection again employed with the same force as before. The blood after a little while began to flow from the pulmonary vein in a continuous stream, though a smaller one than before, and it took between 60 and 70 seconds to fill the measure up to the given point.

These experiments were repeated several times with uniform result.

January 16.—Experiments repeated with the assistance of Mr. Bruce and Mr. Gill. Lungs, those of a dog recently killed.

Experiment 1.—Lungs moderately expanded; pressure of injection 1¾ inch of mercury. Measure filled in thirty seconds.

Experiment 2.—Lungs permitted to collapse by their own elasticity; precisely the same result.

Experiment 3.—Air forced into the jar to pressure of 1 inch of mercury, with same injecting force; measure filled in 40 seconds.

Experiment 4.—On increasing the air-pressure, the bladder gave way at some point, and air began to escape. The tube connected with the trachea was then instantly seized and secured by a ligature, thus preventing any expansion of the lung; the stopcock 5 was then opened, and the air-pressure allowed to become normal. The lung was thus maintained in a state of complete collapse, but without any external pressure. On injecting with the same force as before, it required 37 seconds for the measure to become filled up to the mark.

In August, 1867, a third set of experiments were conducted with lower air-pressures in consequence of the

post-mortem observations above referred to, Mr. Alex. Bruce again kindly assisting. The experiments were performed with one lung only, the left of a sheep, and the blood used was thinned with a little water.

Experiment 1.—Lung expanded moderately; force of injection 1¾ inch of mercury; measure filled in 88 seconds.

Experiment 2.—Lung collapsed by its own elasticity; same injecting force; measure filled in 85 seconds.

Experiment 3.—Lung compressed by air-pressure equal to 9 inches of water; same injecting force; measure filled in 230 seconds.

Experiment 4.—Lung compressed by 5 inches of water; measure filled in 190 seconds.

Experiment 5.—Air tube secured by ligature, and pressure removed; measure filled in 125 seconds.

Inferences from these Experiments.—1. That there is no appreciable difference in resistance to the circulation through a moderately expanded lung, and one collapsed by its own elasticity.

2. That when the lung is more completely collapsed there is a decided increase in the resistance to the circulation through it.

3. That (Exp. 4, second series; Exp. 5, third series) the complete collapse of the lung has a very marked effect in retarding the circulation through it, independently of all external pressure.

In illustration of the effect this pulmonary obstruction has upon the general circulation, an observation of Marcy's may be well quoted. "Un point très important et qu'on ne saurait trop répéter pour le

bien établir, c'est que la même quantité de sang doit, dans un temps donné, traverser les deux appareils circulatoires. Sans cela l'une des circulations recevant à chaque instant un peu plus de sang qu'elle n'en laisse passer, il s'ensuivrait une stase qui irait en augmentant sans cesse et serait incompatible avec la vie."—*Marey, Physiologie Medicale de la Circulation du Sang,* p. 30.

In this way, in cases of pneumothorax, in which the pressure is marked, the normal quantitative relation between the two circulations is interfered with, and death would result, but that the systemic veins admit of some, and the portal system of still greater engorgement, by which a certain amount of the surplus blood is temporarily removed from the general circulation, and time is allowed for more permanent relief either naturally or by treatment.

Neither displacement of heart nor dilatation of the thoracic parietes unless they be very considerable are sufficient to shew the existence of pressure. I have already dealt with the question of cardiac displacement. As regards thoracic dilatation, M. Behier* truly remarks that it is no real distension but simply an absence of retreat. The chest on the side filled by the gaseous collection does not recover itself at the time of expiration, but remains in the position of deep inspiration. M. Behier goes so far as to say that there cannot be any intra-thoracic air pressure even when the opening is valvular, on the ground, that, (1.) air can no longer lift the valve when there is any ex-

* Conférence de Clinique Medicale, 1864.

cess of pressure within tho pleura over that of the atmosphere, and, (2.) he denies the efficacy of a deep inspiration by saying that the deepest inspiration is taken at the moment of rupture and cannot be afterwards exceeded. He further states that (3.) the escapo of air on puncturing the affected side, after death is no evidence of pressure, this being due to the atmospheric pressure exerting itself unopposed upon the depressible parietes of the pleural cavity, maintained no longer by any force in a state of dilatation, also to the elastic recoil of the parietes of this cavity which were distended at the time of inspiration. That there is in some cases of pneumothorax, a very considerable air-pressure within the pleura, I think after the post-mortem experiments in the cases above quoted, cannot be denied; and it seems to me that M. Behier overlooks the true mechanism by which the chest is expanded in these cases. The chest wall, when set free from the elastic traction power of the lung, expands, by virtue of its resilience, to a certain extent, i. e. it arrives, independently of the traction of the lung on the one hand, or the action of the inspiratory muscles on the other, at its *position of rest* and, when at this point of expansion, it exercises no pressure whatever upon the air effused within the pleura. The muscles of inspiration, however, now come into play and expand the chest wall still further, and when this action ceases, there is the same tendency to recoil to its position of rest, this, however, is prevented by the air which entered the pleura during the inspiration but cannot again escapo—so that tho air thus enclosed within the pleural cavity sustains tho

whole force of the resilience of the chest wall. Looking at the great elasticity of the air, the smallness (generally) of the opening in these cases, and the fact that the lung may expand a little at first with inspiration, one would not expect the maximum expansion of the chest, and the maximum pressure, to be attained at once; and, as a matter of fact, it often takes some hours before this point is arrived at. Atmospheric pressure has really nothing to do with the escape of air on puncturing the chest post-mortem: this escape is wholly due to the forcible return of the boundaries of the pleural cavity to their normal state of rest, which had been previously prevented by the accumulation of air.

B. The state of the other lung will obviously affect the symptoms present in Pneumothorax, which will be intense and fatal, *cæteris paribus*, in direct proportion to its disease.

The effect of air in the pleura upon the lung of the opposite side is, as shown in the experiments on the dog and human subject above quoted, to cause its partial collapse, firstly, by removing the resistance to its elastic tension, and secondly, when the pressure of accumulation comes into play, this lung is further embarrassed by the displaced heart and direct pressure.

The exchange of a certain negative pressure which is exercised upon the cavities of the heart, and doubtless aids considerably its refilling after systole, for a more or less positive air-pressure in pneumothorax, must tend to embarrass its action in some degree. In Case 8, where there was a pressure on one side of 3½, and on the other

of 2·7 inches of water, the cavities of the heart were found empty, with the exception of half an ounce of blood in the left ventricle. The direct pressure to which the heart was exposed in this case may very probably have prevented the return of blood into the right cavities.

The nature of the opening will affect the symptoms, which will be the more urgent in proportion as the opening is more or less completely valvular. It is quite obvious that an opening may be potentially valvular which does not appear to be actually so post-mortem.

The previous condition of the patient is much in accordance with that of his lungs.

The peculiar temperament of the patient will greatly influence the amount of shock he will experience on being seized with pneumothorax.

The *prognosis* in a case of pneumothorax, always very grave, rests chiefly upon a knowledge of the previous condition of the patient, the amount of disease present in the other lung, and the probable valvular or non-valvular nature of the opening. It is, of course, special to each case, but it it may be laid down as a general rule that if the opening be valvular the patient will die within a few hours unless relief be afforded by paracentesis; whereas if the opening be non-valvular (indicated by amphoric respiration and more or less movement of chest wall), the patient may survive for weeks, months, or even years, dying, in fact, either from exhaustion from empyema, or from the progress of the disease in the other lung. (a).

(a) Of course those cases must be excluded which are immediately fatal from collapse of the only remaining portion of healthy lung.

The *treatment* of a case of pneumothorax will vary according to the manner in which life is threatened, whether by asphyxia, shock, or exhaustion.

In order that we may have the best chance of obviating the tendency to death from asphyxia, it is necessary to bear in mind the significance of the physical signs present, and the mechanism by which the pressure is produced. If there is amphoric respiration audible, the opening will be free, and there is no danger of death from pressure. Rest should be secured to the affected lung as far as possible, to give the opening in the pleura an opportunity of becoming closed. This object will best be attained by the application of a broad piece of strapping round the affected side, and reaching a little beyond the median line both in front and behind; by this means the action of the respiratory muscles on the affected side is restrained without interfering with respiration on the healthy side. If however the opening be valvular, life is threatened by asphyxia from accumulation of air in the pleura and consequent impediment to the action of the heart and the expansion of the opposite lung; evidence of this condition is found in the complete absence of respiration, and the increasing urgency of the symptoms. I have already endeavoured to shew that the pressure in these cases is due to the expansion of the parietes by the action of the inspiratory muscles beyond their position of mean rest, and their forcible tendency to return to this position. If then we suspect the presence of a valvular opening early enough, and apply a broad piece of plaster accurately round the affected side, as above

suggested, we may by restraining the action of the inspiratory muscles upon the ribs, altogether prevent the occurence of intra-pleural pressure. The diaphragm can take no active part in increasing the pressure of the enclosed air, because directly it becomes flattened or concave towards the thoricic cavity, it can no longer exercise any inspiratory power, so further accumulation by this means cannot take place. It has been said by some observers,* that the diaphragm may become concave towards the thorax and its action reversed, thus with the inspiratory effort diminishing instead of increasing the capacity of the chest on the affected side. Its action is however more probably altogether paralysed. Any abdominal distension from flatus should be guarded against.

In all cases in which death is imminent from asphyxia, puncture of the chest should be practised, if the other lung do not present signs of such extensive disease as to render all interference hopeless.

It is still a question what kind of opening is most suitable—whether a small opening or a free one. The latter would give the lung the most complete rest, and, with the other lung tolerably sound, and the assistance of the valuable disinfecting agent carbolic acid as a lotion, might be tried with success. I should recommend, however, a valvular opening made with a small trochar, and the subsequent application of the plaster to the side as above directed.

Purgatives.—The way in which relief is naturally afford-

* Dr. Duncan. *Edin, Med. and Surg. Journ.* Vol. 28, 1827.

ed to the circulation in pneumothorax and in obstruction of the pulmonary system of vessels from other causes, is, by engorgement of the venous system, and more particularly of the portal system, the veins of the latter having no valves. The cautious administration of such purgatives, therefore, as will produce a watery flow from the intestines, and are not depressing, will be useful. The tartrates of soda and potash, the sulphate of magnesia, decoction of aloes, etc., with some stimulant, may be recommended.

If the systemic veins should be much engorged, and the dyspnœa great, bleeding will give relief, but cases in which this treatment is necessary must be very rare, and limited to those occurring in persons of good, or even robust general health. Dry cupping freely employed is often of great service.

Opiates are extremely useful to relieve the shock, to calm the patient, and to check that dyspnœa which is not mechanical, and the subcutaneous injection of morphia is a convenient mode of administration.

Stimulants may be absolutely necessary at the moment of attack to avoid death from syncope, but if no immediate danger from this cause be present, they had better be given sparingly. Opium, indeed, is the best stimulant in these cases.

An abstract is here given of those cases above referred to in which the pressure within the chest has been tested post mortem, and only such brief notes are added as appear necessary to explain the peculiarities of each case.

Case 1.—A patient who died at the Chelsea Home. (No notes taken of the case except the pressure.) Trocar inserted below the nipple. Pressure=4 inches of water.

Case 2.—S. T., aged 22, male, out-patient at Brompton Hospital, under Dr. Sanderson. Ill eight months with noisy cough, dense expectoration, emaciation, night sweats. Hæmoptysis. Condition of chest:—Consolidation of upper third of right lung. Seized on June 18 with sudden dyspnœa and well-marked symptoms and signs of pneumothorax on the right side. Died June 20. Autopsy:—Trocar thrust in at the fifth interspace. Pressure=5 $\frac{3}{10}$ inches of water. Two perforations found in the lateral and upper part of superior lobe of right lung communicating with small cavities. Three-quarters of a pint of turbid fluid in the pleura. Some consolidation and softening of left lung. There was no decomposition present nor distension of the abdomen.

Case 3.—S. F., aged about 25, female, out-patient at Brompton Hospital, under Dr. Powell. Ill some time with troublesome cough, but chief suffering latterly had been from relaxed bowels, with tympanites and frequent vomiting and eructations. Condition of chest: Moderate amount of softening at both apices. Seized on July 15 during a violent fit of retching with severe pain in the left side and much dyspnœa. Marked hyper-resonance extending over the cardiac region; respiration very distant, feeble. Heart's impulse not perceptible to touch; detected by the stethoscope to be displaced to the right. Posteriorly, hyper-resonance, blowing amphoric respiration at angle of scapula, with some metallic tinkling. Died in a few days. Pressure within the chest tested=2 inches of water; no further examination made. This patient after the first attack did not suffer greatly from dyspnœa; died from exhaustion from diarrhœa and vomiting, which resisted all treatment. The well-marked amphoric breathing indicated a tolerably free communication.

Case 4.—J. P., aged 44, male, in-patient at Brompton Hospital, under Dr. Sanderson. An ordinary case of phthisis. On admission, consolidation and softening of both sides were observed; some cavernous rhonchi at the left apex. Doing well until July 18, when, while walking in the grounds, he felt some pain of a cutting character in the right side. He went in shortly afterwards, and on examination a few moist superficial râles were heard in the right axilla at the seat of pain. There was no dyspnœa at this time. Dyspnœa came on gradually, and increased steadily until death. Respiration distant, not amphoric in quality. Died July 20. Pressure tested=3 ¾ inches of water. Perforation in left lung at outer and posterior part of upper lobe, 3 inches from apex, perfectly valvular, a slit a quarter of au inch broad communicating with a slanting canal running through the thickened pleura and terminating by a rounded opening in a small cavity. Right lung infiltrated with tubercle; a small cavity at apex. This case was remarkable for the absence of shock, the few physical signs present, and the rapid destruction of the patient.

Case 5.—T. W., aged 23, male, in-patient at Brompton Hospital, under Dr. Sanderson. Admitted suffering from cough, expectoration, dyspnœa, etc.; cavities at both apices, with softening below on right side. Seized on October 6 at 12.30 a.m., when he was found lying in bed rolling about in intense agony. Countenance dusky; pulse quick, small; respiration very frequent and shallow; extreme tenderness on percussion at right anterior base, where the pain was most intense, with hyperresonance, and some distant tubular breathing. Died October 7 at 7 a.m. Autopsy: Trocar inserted at fifth right interspace. Pressure=2 inches of water. Opening not found, the lung having been cut into. Base of right lung collapsed. This was the only portion of lung which was not extensively diseased. Left lung extensively diseased throughout.

Case 6.—C. M., aged 34, male, in-patient at Brompton Hos-

pital, under Dr. Quain. Admitted as an ordinary case of phthisis, with double cavities and extensive disease of both lungs. Seized on the evening of March 29 with sudden pain in the right side and great dyspnœa. Right anterior and axillary base resonant; respiration inaudible; much creaking and friction sound, with some fine rhonchus. Dyspnœa increased, and side became bulged and hyper-resonant. Died on the morning of March 31. Autopsy: Pressure=4 inches of water. At lateral part of right lung three inches below the apex, there is a circular opaque patch, forming one side of a superficial flattened cavity, at the apex of which is a small slit. The thin pleural side forms a very complete valve. Lung compressed and tuberculised throughout. Large cavity at apex of left lung, which is tuberculised throughout. Right cavities of heart somewhat dilated. Pulmonary artery measures across valves 5 and three-eighths of an inch; left cavities nearly empty; aorta measures 3 and one eighth; liver large (58 ounces); much hepatic congestion; spleen not enlarged. The absence of amphoric respiration, with very valvular opening, were well illustrated here. Paracentesis was useless from extensive disease of the lung. (Vide *Transactions of the Pathological Society*, Vol. xix, p. 77.

Case 7.—G. H., aged 17, male, in-patient at Brompton Hospital, under Dr. Quain. Autopsy: Pressure = 1 ¼ inch. Two openings at base of the left lung communicating with small cavities. Lung much diseased. Pericardium contained air, which had entered from pleura through an opening apparently congenital. Other lung considerably diseased. (a) In this case the openings were free and the pressure very slight.

Case 8.—R. B., aged 24, female, in-patient at Bromptom Hospital, under Dr. Pollock. On admission in June, 1868, suffering from moderate dyspnœa, with some lividity, but able

(a) For further notes is this case, see *Pathological Trans.* Vol. XX. p. 99.

to walk about. Expansion fair on both sides, less so on left; no bulging on left side; hyper-resonance to fourth rib, with dulness below that level anteriorly and posteriorly; amphoric respiration in scapular region. Succussion signs, etc., obtained. Cardiac impulse to right of median line; apex at ensiform cartilage. Signs of effusion increased, leading to considerable dyspnœa. Patient died suddenly July 1, 1868, with signs of perforation of opposite lung. Pressure left side = 3·5 inches of water; of right side = 2·7 inches of water. Left pleura contained two quarts of pus. An aperture the size of a sixpence existed at the apex of lung, communicating with a large cavity, the walls of which were very thin. Right pleura distended with air. There was a small aperture at apex communicating with a small cavity which itself communicated by means of a narrow sinus with a larger cavity. Heart healthy; cavities empty, except about half an ounce of blood in the left ventricle. When patient was first seen about three weeks before death, it was concluded from the amphoric breathing and absence of pressure-signs, that the displacement of the heart was simply the result of partial collapse of the opposite lung. The pressure found post mortem on the left side was no doubt due to the later accumulation of fluid rendering the opening practically valvular. The pressure of the opposite side was the result of the valvular opening, and though the patient only survived ten minutes, it had risen to 2·7 inches. Post-mortem was made by Mr. Gill, Resident Clinical Assistant.

I have not considered in the present pamphlet cases of local pneumothorax in which the air is confined by adhesions to a limited portion of the pleural cavity. These cases are not very uncommon and are often overlooked. They usually closely resemble large superficial lung cavities in physical signs, and require no special treatment.

General pneumothorax is not unfrequently local at first, the adhesions becoming rapidly torn down, and this tearing down of the adhesions is no doubt the chief cause of the acute pain which accompanies the seizure.

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